Effects of Growing Season and Fungicide Type on the Development of *Alternaria solani* and on Potato Yield

D. Shtienberg and **D. Blachinsky,** Department of Plant Pathology, ARO, The Volcani Center, P.O. Box 6, Bet Dagan 50250, Israel; and **G. Ben-Hador** and **A. Dinoor,** Department of Plant Pathology and Microbiology, The Hebrew University of Jerusalem, Faculty of Agriculture, P.O. Box 12, Rehovot 76100, Israel

ABSTRACT

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The intensity of early blight disease caused by *Alternaria solani* and its effects on yield were evaluated in potato crops in the autumn (four experiments), winter (one experiment), and spring (five experiments) growing seasons in Israel. Analysis of disease progress curves revealed that early blight is more important in the autumn and winter than in the spring in the northern Negev region of Israel. Similarly, differences in yield between fungicide-treated and nontreated plots were significant ($P < 0.05$) in four of the five autumn and winter experiments (20.6 to 24.0% increase) but in none of the spring experiments. In the autumn and winter seasons, early blight did not affect numbers of tubers produced per unit area but reduced tuber weight. The efficacy of the systemic fungicides tebuconazole and difenoconazole against *A. solani* was compared with that of the common protectant fungicides chlorothalonil and mancozeb. Tebuconazole significantly decreased lesion expansion rate and chlorothalonil did not. In some of the autumn and winter experiments, but in none of the spring experiments, the systemic fungicides were significantly more effective than the protectants in suppression of *A. solani*.

Additional keywords: *Solanum tuberosum*, yield loss assessment

Early blight, caused by *Alternaria solani* Sorauer, is a major fungal pathogen of irrigated potato (*Solanum tuberosum* L.) crops in hot climates. *A. solani* is an airborne pathogen, the dark, multicellular spores of which are dispersed by wind, rain, and overhead irrigation splash. Germ tubes penetrate the leaves directly or through wounds. On foliage, characteristic symptoms appear as dark, concentric rings of necrotic tissue (16). The response of potato to *A. solani* changes with age: immature plants are relatively resistant to early blight but, after tuber initiation, susceptibility increases gradually, and mature plants are very susceptible to the pathogen (12,15, 16). Thus, early blight is principally a disease of senescing plants (23). Some authors reported that severe epidemics reduce yields by up to 30% (e.g., 2,5,6,18). Early blight can be controlled with fungicides (23) but the relationship between disease suppression and the amount of yield increase is unclear (e.g., 1,3,15).

Corresponding author: D. Shtienberg
E-mail: dani@agri.huji.ac.il

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Potatoes are grown in Israel (30° E, 31° N) in several regions. The main area of production (4,200 ha, 60% of the national cultivated area) is in the northern Negev. The climate there is semiarid with mild winters and hot, rainless summers. There are two main seasons for potato production, autumn and spring, and one minor season, winter (9). For the autumn crop, potatoes are planted in late August to early September and harvested during December and January. For the spring crop, potatoes are planted in late January to February and harvested during late May, June, and July. The few winter season potatoes are planted in October and November and harvested during late March, April, and early May. The growing seasons differ markedly in respect to the environment (Fig. 1): for the autumn crop, growth starts when temperatures are high and the days are relatively long, and continues under decreasing temperature, day length, and radiation. For the spring crop, growth starts when temperatures are relatively low and days are relatively short, and continues under increasing temperature, day length, and radiation. For the winter crop, temperatures, day length, and radiation are relatively low throughout the entire growing season (9).

Reports are contradictory on the intensity of early blight and its effects on yield during the different growing seasons in Israel. Nachmias et al. (11) found that symptoms of *A. solani* were more severe and yield losses were higher in the spring than in the autumn, whereas Rotem (15,16) reported greater disease and yield losses in the autumn.

To suppress early blight, growers often apply fungicides to the foliage, five to 10 times during typical autumn and winter growing seasons and three to eight times during spring. Until recently, only protectant fungicides (e.g., maneb, mancozeb, and chlorothalonil) were available for suppression of *Alternaria.* The systemic fungicides tebuconazole (Folicur) and difenoconazole (Score) are now available for use on potato. In the case of cotton, these systemic fungicides were effective against *A. macrospora* for a longer period and in some cases provided superior disease suppression and higher yields when compared with protectant fungicides (20,21). The efficacy of the systemic fungicides in suppressing potato early blight had not been examined thoroughly prior to the beginning of this study.

In this study, intensity of *A. solani* and its effects on yield were evaluated in potato crops grown in autumn, winter, and spring to identify the impact of early blight epidemics in those seasons. This information is important for epidemiological purposes and essential for disease management decisions. In addition, the efficacy of the systemic fungicides tebuconazole and difenoconazole for control of *A. solani* was examined. Some of the results have been published (19).

MATERIALS AND METHODS

Field studies. Experiments were conducted in commercial fields located in the northern Negev region of Israel during the years 1989 to 1995. Certified potato seeds (whole tubers, each weighing 50 to 100 g) were planted during the first half of Sep-

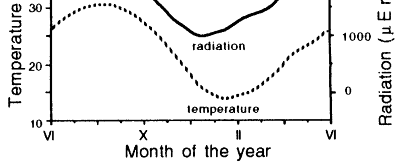

Fig. 1. Long-term average maximal temperature and radiation intensity in the northern Negev region in Israel. Periods of potato production are indicated by arrows.

tember in 1989, 1990, 1992, and 1993 for the corresponding 1989 to 1990, 1990 to 1991, 1992 to 1993, and 1993 to 1994 autumn seasons; during the second half of February in 1990, 1991, 1994 (two trials, 1994-I and 1994-II), and 1995 for the corresponding spring seasons; and in early November 1994 for the corresponding 1994 to 1995 winter season. Cultivar Alpha was planted in most experiments. In spring 1994, cvs. Desiree and Draga were planted, and in winter 1994 to 1995, cv. Nicola was planted. All cultivars are susceptible to *A. solani*. The crops were irrigated via overhead sprinklers (once every 3 to 5 days) for a total of 5,000 to 6,000 m^3 water/ha in the autumn and winter seasons and 6,000 to 7,500 m^3 water/ha in the spring season. A total of 300 to 350 kg N/ha was applied to each crop, 80 to 150 kg N/ha of which was applied prior to planting and the rest during crop growth by means of the irrigation system. Fertilization was halted about 30 days before vine kill. Vines were killed chemically 120 to 130 days after planting (dap) in the autumn season, 180 dap in the winter season, and 140 to 150 dap in the spring season.

The experiments were laid out in randomized block designs with four (or five in autumn 1990 to 1991, winter 1994 to 1995, spring 1991, and 1994-I) replicates per treatment. Plots consisted of four 7-m-long rows. Inter-row spacing was 0.96 m and intra-row spacing of plants was 25 to 30 cm. Plots were separated from each other by fallow areas approximately 1 m wide. Experimental plots were not inoculated artificially. Inoculum was present naturally at the test sites. In each trial, one protectant and one systemic fungicide were used. Protectant fungicides were mancozeb (Manzidan, 80WP, Rohm & Haas, Croydon, UK) at a rate of 2.4 kg a.i./ha or chlorothalonil (Bravo, 50% SC, ISK Biotech, Painesville, OH) at a rate of 1.5 kg a.i./ha. Systemic fungicides were tebuconazole (Folicur, 25% EC, Bayer AG, Leverkusen , Germany) at a rate of 0.25 kg a.i./ha or difenoconazole (Score, 25% EC, Ciba AG, Basle, Switzerland) at a rate of 0.25 kg a.i./ha. Fungicides (in 260 to 300 liters of water per ha) were applied by means of a motorized backpack sprayer at a pressure of 275 kPa with cone-jet X6 nozzles.

Each experiment included 6 to 12 different treatments (spraying schedules). These treatments differed in time of spraying initiation, spraying intervals, and type of fungicide (protectant, systemic, or integration of protectant and systemic fungicides). Disease progress curves and harvested yields are given only for three treatments that were included in all experiments. Results of the other treatments were analyzed but they are included only in the yield loss section (see below). The three common treatments were as follows: (i) nontreated control; (ii) protectant fungicide (chlorothalonil or mancozeb) applied on a 7-day schedule, with a total of 8 to 12 sprays; and (iii) systemic fungicide (tebuconazole or difenoconazole) applied on a 14-day schedule, with a total of 4 to 6 sprays. Spraying against early blight was initiated soon after the appearance of visible *A. solani* lesions in the canopy, but not before tuber initiation (i.e., tubers less than 3 cm in diameter). This occurred 40 to 60 dap in autumn, 60 to 80 dap in spring, and 108 dap in the winter season.

Disease assessment. Disease was assessed visually by two individuals independently and the scores were averaged. Assessments were made every 7 to 14 days from the first appearance of early blight symptoms until the end of the season (vine kill). Disease severity (i.e., the proportion of plant necrosis primarily from infection by *A. solani,* but including natural senescence) in the two middle rows of each plot was estimated with an assessment system described by Fry (4). In the early stages of the epidemics, the only cause for foliar desiccation was *A. solani,* so it was possible to assess disease severity accurately. Since *A. solani* develops primarily on older leaves and disease severity increases as the crop ages, it was not always possible at later stages of the epidemic to distinguish between necrosis induced by the pathogen and that resulting from natural senescence. Therefore, when assessing disease severity, no attempt was made to identify specific causes; the score represented only the portion of necrotic foliage. Disease scores were used to calculate the area under the disease progress curve (AUDPC) for each of the treatments (6). Thus, an indication of the sole effect of early blight was obtained by examining the difference in AUDPC between fungicide-treated and nontreated plots.

Yield loss assessment. After crop maturity, tubers from a 5-m^2 area were harvested from the center of each experimental plot of all treatments. Tubers were collected by hand and weighed, and the yield per hectare calculated. The difference in yield (t/ha) between the fungicide-treated and the nontreated plots was determined for all treatments in the experiments.

The effect of early blight on yield was quantified for each season by formulating the disease severity/yield loss relationship. For disease severity, differences in AUDPC between fungicide-treated and nontreated control plots were used. For yield loss, differences in yields between fungicide-treated and nontreated control plots were used. A similar approach was used previously by Johnson and Teng (7).

The effect of *A. solani* on yield accumulation was examined in two experiments conducted in the autumn (1992 to 1993) and winter (1994 to 1995). In both experiments early blight was severe. Tubers were collected three times during the bulking period from a 5-m^2 area in each experimental plot of a tebuconazole spray treatment and of the nontreated control. In the 1992 to 1993 experiment, harvests were 106, 128, and 155 dap. In 1994 to 1995, harvest was 160, 181, and 195 dap. The number of tubers in each harvest and overall weights were used to calculate average tuber weights. Results of all experiments were subjected to analysis of variance and when the *F* values showed significant differences, Fisher's protected least significant difference test was applied at $P = 0.05$.

Efficacy of the systemic fungicides. Effects of the protectant fungicide chlorothalonil and the systemic fungicide tebuconazole on lesion expansion rate (LER) were determined in trials conducted during the autumn seasons of 1989 to 1990 and 1990 to 1991 and the spring seasons of 1990 and 1991. Leaflets with *A. solani* lesions were marked with red, numbered, plastic bands stamped across the petioles. Leaflets were selected on the upper parts of the plants and only if there was just one lesion in an area smaller than 10 mm^2 on that leaflet. Changes in lesion area were measured at 5- to 7-day intervals. Nondestructive measurements were performed by comparing the shape of the lesions with predetermined standard diagrams on a transparent slide. The standard diagrams included drawings of 20 lesions with different shapes and sizes; shapes were similar to those typical of *A. solani* and sizes ranged from 10 to 314 mm^2. Measurements of individual lesions continued until (i) the leaflet senesced, (ii) it was no longer possible to identify the original lesion, (iii) the observed lesion reached the margins of the leaflet, or (iv) secondary lesions interfered with its growth. Four lesions were monitored on plants in each experimental replicate of the protectant fungicide chlorothalonil, the systemic fungicide tebuconazole, and the nontreated control. Measurements were performed 70 to 110 dap in the autumn seasons and 80 to 120 dap in the spring. LER was calculated for each lesion separately by regressing the natural logarithm of lesion area for each sampling date over time. The slope of the regression equation was used as an estimate of LER. Then the mean LER was calculated for each treatment. The approach for calculating LER is similar to the one used previously by Johnson and Teng (8) and Shtienberg et al. (21).

RESULTS AND DISCUSSION

Seasonal effects. Onset of early blight varied substantially among the three growing seasons. In the autumn crops, it occurred 60 to 70 dap; in spring crops, 70 to 90 dap; and in the winter crop, 115 dap. Disease progress curves typical of autumn and spring crops showed different patterns (Fig. 2). In all the autumn and winter seasons, AUDPC values for fungicide-treated plants were significantly less than those for nontreated plants. By contrast, spraying did not reduce AUDPC significantly below nontreated control in three of the five experiments conducted in the spring season (Table 1). Mean control for experiments conducted in the autumn and winter was 47.9 to 60.2%, compared with 15.9 to 29% for experiments conducted in the spring.

In four of the five autumn and winter experiments, differences in yield between fungicide-treated and nontreated plots were significant (Table 1). Mean yields were 26.2 t/ha in nontreated plots, 31.6 t/ha (20.6% increase over nontreated) in plots treated with the protectant fungicides, and 32.5 t/ha (24.0% increase over nontreated) in plots treated with the systemic fungicides. In none of the five spring experiments was yield increase significant following fungicide treatment.

The relationships between the difference in AUDPC of fungicide-treated and nontreated plots, and between the differences in yields of the same treatments, were positive and significant in the autumn and winter seasons, but not in the spring (Fig. 3). This difference is the outcome of both disease pressure and the efficacy of the fungicide applied to the foliage. Since AUDPC is the integration of disease severity over time, differences in AUDPC between fungicide-treated and nontreated plots reflect the contribution of the spray treatment to extending the green leaf area duration. This supports Rotem's conclusion (15,16) that early blight is more important in autumn and winter crops than in spring crops. The variable effect of the disease in the three growing seasons may be a reflection of the different seasonal patterns of environmental conditions such as temperatures and radiation. Such differences could be important particularly during tuber bulking. Temperature and radiation are relatively low toward the end of crop growth in the autumn and winter seasons (Fig. 1); thus, further development of the foliage is restricted. In spring crops, on the other hand, temperature and radiation are higher (Fig. 1), making foliage senescence relatively slow, and giving ample opportunity to the plant to produce new leaves and overcome the effects of the disease (18,22).

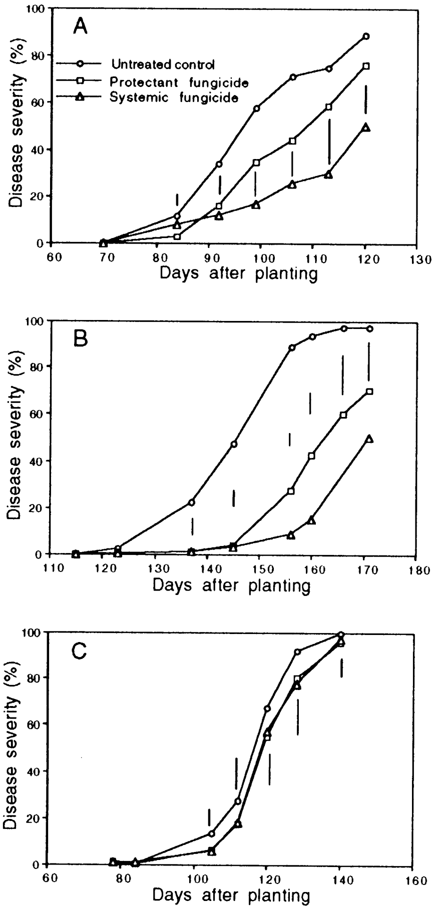

Fig. 2. Effects of growing season and fungicide type on plant necrosis primarily from infection by *Alternaria solani*, but including natural senescence. Mancozeb (protectant fungicide) and tebuconazole (systemic fungicide) were applied on a 7- and a 14-day schedule, respectively. Data are from the **(A)** 1992 to 1993 autumn season, **(B)** 1994 to 1995 winter season, and **(C)** 1994-I spring season. Bars indicate the least significant difference values ($P < 0.05$) for each sampling date.

Sandstorms occur more frequently in the autumn and winter than in the spring. Sandstorms cause visible damage to foliage as well as small, invisible wounds. The sequence of increased (i) dispersal of spores during sandstorms, (ii) susceptibility of foliage damaged by such storms, and (iii) dewfall after the storms, promotes early blight development and is associated with severe disease outbreaks (13,14,16).

The effect of early blight on yield accumulation was evaluated in two experiments (Figs. 2 and 4). The difference in tuber weight between fungicide-treated and nontreated plots was insignificant 106 dap, when disease severity in the former treatment was 25.5% and in the latter, 71.2%. Two weeks later, disease severity was 50.0 and 88.7% in these two treatments, respectively, and the difference in tuber weight (17 g per tuber or 16.2%) between the treatments was significant. Up to physiological maturity (155 dap), tuber weight in the nontreated plots did not change because the foliage was completely destroyed, but tuber weight in the fungicide-treated plots increased and the difference between fungicide-treated and nontreated plots was 25 g per tuber (23.8%). The number of tubers produced per unit area did not differ significantly among the treatments on any assessment date. Since similar patterns were observed in the 1994 to 1995 winter experiment, results are not shown.

In a previous study it was demonstrated that early blight may reduce both the rate of tuber bulking and the duration of yield

Table 1. Effects of growing season and type of fungicide on the area under the disease progress curve (AUDPC) and on potato yield[x]

Growing season	Year	Fungicide treatment[y]	AUDPC	Yield (t/ha)
Autumn	1989 to 1990	Untreated	3.3 a[z]	35.4 a
		Protectant (C)	1.6 b	36.3 a
		Systemic (T)	1.5 b	37.1 a
	1990 to 1991	Untreated	15.6 a	17.3 a
		Protectant (C)	9.6 b	22.8 b
		Systemic (T)	7.6 b	27.4 b
	1992 to 1993	Untreated	20.1 a	22.4 a
		Protectant (M)	13.8 b	27.9 b
		Systemic (T)	7.9 c	30.5 b
	1993 to 1994	Untreated	16.7 a	34.2 a
		Protectant (M)	8.3 b	42.9 b
		Systemic (D)	10.6 b	38.7 ab
Winter	1994 to 1995	Untreated	26.3 a	21.6 a
		Protectant (M)	9.4 b	27.9 b
		Systemic (T)	5.0 c	28.8 b
Spring	1990	Untreated	16.2 a	25.4 a
		Protectant (C)	15.7 a	26.8 a
		Systemic (T)	13.4 a	28.8 a
	1991	Untreated	10.3 a	35.9 a
		Protectant (C)	6.9 b	38.2 a
		Systemic (T)	6.6 b	39.3 a
	1994-I	Untreated	13.2 a	36.9 a
		Protectant (M)	9.6 a	40.6 a
		Systemic (T)	13.9 a	36.6 a
	1994-II	Untreated	4.7 a	31.2 a
		Protectant (M)	2.3 a	34.5 a
		Systemic (D)	2.4 a	32.7 a
	1995	Untreated	9.6 a	52.0 a
		Protectant (M)	3.8 b	50.0 a

[x] Disease was the result of infection by *Alternaria solani* and of natural senescence.

[y] Protectant fungicides: C = chlorothalonil; M = mancozeb. Systemic fungicides: T = tebuconazole; D = difenoconazole.

[z] In each experiment, numbers followed by a common letter do not differ significantly ($P < 0.05$) as determined by the least significant difference test.

accumulation (18). Tubers are initiated over a period of approximately 2 weeks at the beginning of the reproductive phase (40 to 80 dap, depending on the cultivar and the growing conditions), at which time the host is relatively resistant to *A. solani*. Final yield depends on the rate of bulking and the length of time over which it takes place. Tuber growth ceases completely with death of the foliage (10).

Efficacy of the systemic fungicides. LERs on plants with either protectant or systemic fungicides were examined in the autumn and spring seasons. Since results of experiments conducted in each season exhibited similar trends, and the variance was homogeneous, data were pooled. In general, LER was more rapid in the spring than in the autumn, probably due to the higher temperatures in the former. Chlorothalonil did not affect LER significantly, but tebuconazole significantly decreased LER compared with the rate calculated for the nontreated plots (Fig. 5).

Comparison of the AUDPC in four experiments in the autumn, one in the winter, and five in the spring season, enables us to identify the relative efficacy of protectant and systemic fungicides against early blight. The AUDPC in plots treated with the systemic fungicides was significantly lower than that in plots treated with protectant fungicides in two of five experiments conducted in the autumn and winter seasons. Mean AUDPC values were 16.4 (nontreated control), 8.5 (protectant fungicide), and 6.5 (systemic fungicides). Differences in AUDPC between the protectant and systemic treatments in the spring season were insignificant in all trials. Mean AUDPC values for these treatments were 11.1 (nontreated control), 8.6 (protectant fungicide), and 9.1 (systemic fungicides) (Table 1). The results indicate that management of early blight in the autumn and spring seasons is essential and may lead to significant yield increases, but the need for its suppression in the spring season is questionable.

Results of these experiments with potatoes resemble those obtained with cotton and *A. macrospora* (17,20). In both pathosystems the systemic fungicides applied on a 14-day schedule were in some cases more effective than the protectant fungicides applied on a 7-day schedule. Since the systemic fungicides are more expensive than the protectant, and since it is important to reduce the risk of development of an *A. solani* population resistant to the systemic fungicides, their use should be limited. One way to achieve this goal is to integrate their use with other control measures. Concepts for integration of genotype resistance, age-related resistance, and fungicides (protectant and systemic) were developed recently (21). Application of fungicides is not needed in plants at the vegetative stage, when they are relatively resistant. Accordingly, spraying should be initiated only when host response to *Alternaria* shifts toward increased susceptibility, i.e., at the initiation of the reproductive stage. Frequency of subsequent sprays should be determined according to the genotype resistance of the cultivar and the efficacy of the fungicide, in relation to changes in age-related resistance. Accordingly, protectant fungicides should be applied initially at relatively long intervals, and subsequently at shorter intervals as the crop ages. Toward the end of the season, more effective control, by means of a systemic fungicide, is recommended. These concepts were evaluated and corroborated recently under field conditions for cotton and potatoes (19).

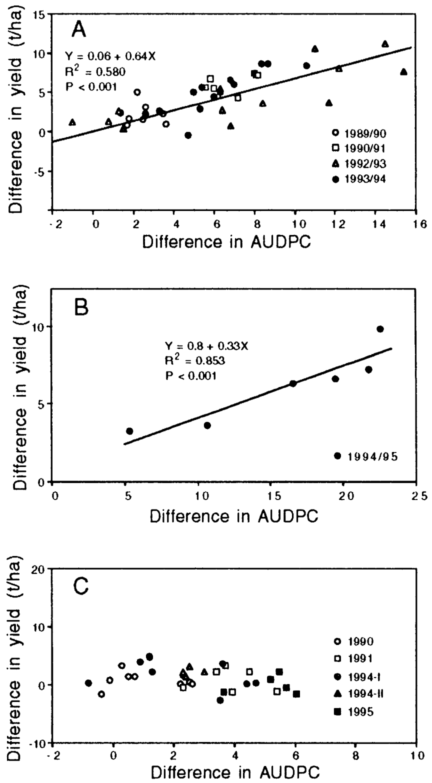

Fig. 3. Relationship between the difference in disease severity (in terms of area under the disease progress curve units) in fungicide-treated and nontreated plots and between the difference in yield between the corresponding treatments in (A) autumn, (B) winter, and (C) spring potato-growing seasons. Disease severity was the result of infection by *Alternaria solani* and of natural senescence.

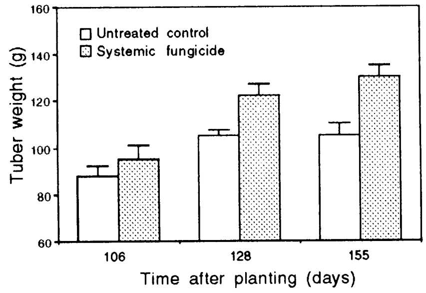

Fig. 4. Effects of the systemic fungicide tebuconazole on changes in potato tuber weight over time, in the 1992 to 1993 autumn season. Bars indicate the SE.

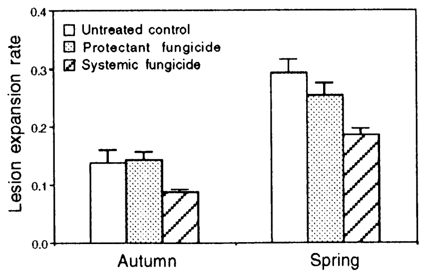

Fig. 5. Effects of the protectant fungicide chlorothalonil and the systemic fungicide tebuconazole on lesion expansion rate ([ln mm^2]/day)of *Alternaria solani* in two growing seasons. Bars indicate the SE.

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